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SUMMARY

The effect of exercise and sleep loss on sleep was studied in four groups of young, physically fit, well-trained U.S. Marine Corps male volunteer subjects. In the first study, Study 1, ten pairs of Marines were observed. One member of each pair was assigned to an "exercise" condition and walked on a treadmill in full combat gear at a speed that induced an elevated heart rate corresponding to 30% of the individual's VO_2 max. Exercise periods occurred during the first half-hour for each of two 17-hr long periods of continuous work (CW). These two CW episodes, designated CW1 and CW2, were separated by a 3-hr nap. Individuals assigned to the exercise condition spent the second half of each hour in the CW period performing cognitive tasks at a computer terminal. The other member of each pair of subjects was assigned to a non-exercise "control" condition. The control subjects performed the same cognitive tasks as the subjects in an exercise routine, but did not walk on the treadmill. Study 1 consisted of two groups: (1) "Nap/Exercise", and (2) "Nap/No Exercise." Study 2 had eight pairs of Marines; the experimental protocol was identical to Study 1 with the exception that neither the exercising subject nor the control subject were permitted to nap between CW1 and CW2. In Study 2 the groups were called: (1) "No Nap/Exercise", and (2) "No Nap/No Exercise". Our results showed that sleep loss increased Slow Wave Sleep (SWS manually scored) duration, and EEG slow wave amplitude, (computer measured), as well as slow wave time during recovery sleep following the CW2. When subjects were totally sleep deprived (Study 2), exercise increased stage 4 and percent SWS. The observed interaction of sleep loss and exercise on SWS was, however, not strong, and requires further experimental confirmation. The working hypothesis of exercise-induced SWS was only partially accepted in this study.

1. Introduction

As a part of a larger study involving simulation of a 5-day, U.S. Marine reconnaissance mission (Englund, Naitoh, Ryman and Hodgdon, 1983; Naitoh, Englund and Ryman, 1982; Naitoh, Englund and Ryman, 1983; see West, 1967) physically fit U. S. Marine Corps volunteer subjects performed cognitive and psychological tasks which were run continuously for up to 45 hours with little or no sleep. The primary purpose of these studies was to measure the "restorative effect" of short sleep,`naps` (Naitoh, 1981; Naitoh et al., 1982). Naps are expected to refresh subjects from the fatigue of continuous work, and thus maintain of effectiveness in cognitive and physical performance.

However, this series of studies yielded additional information about whether sleep deprivation would interact with physical exercise to produce a greater impact on sleep after a mission completion, i.e. recovery sleep, than the impact expected from the effects of sleep loss or exercise alone. The effect of sleep loss combined with physical work on electroencelphalographic (EEG) sleep patterns has been studied by two groups who reported inconsistent results: Webb and Agnew (1973) and Moses, Lubin, Naitoh and Johnson (1977). Recent studies by Horne and Pettitt (1984), Angus, Heselgrave and Myles (in press) and Englund, Ryman, Naitoh and Hodgdon (submitted for publication, examined the combined effect of sleep loss and physical work on cognitive and physical performance, but these studies did not include evaluation of EEG sleep patterns.

Oswald (1980) hypothesized that Slow Wave Sleep (SWS, stages 3 and 4 combined) represented a period of body tissue restoration from fatigue of daily energy expenditure. Greater than normal exercise in a day, as experienced by the Marines in our study, would mean a greater expenditure of physical energy. Hence, Oswald`s restorative theory of sleep would predict a SWS increase during recovery sleep for our exercised Marines. Horne (1981) prepared an extensive review of the literature on the effects of exercise on sleep. Some studies cited in his review gave convincing evidence of an increased SWS after vigorous exercise: e.g., Shapiro (1982) felt that "sleep, particularly SWS, constitutes an active recovery process after the net catabolism of the day".

Slow Wave Sleep has also been found to be increased during recovery sleep after sleep deprivation. (Berger and Oswald, 1962; Williams, Hammack, Daly, Dement and Lubin, 1964; Webb and Agnew, 1977; Moses et al., 1977; Borbely, Baumann, Brandeis, Straugh and Lehmann, 1981). If increased SWS represented

increased fatigue (or uncompensated catabolism), fatigue due to sleep deprivation would be additive to fatigue of physical exercise, resulting in a greater degree of fatigue. Bonnet (1980) suggested that physical exercise alone produced changes in mood and performance similar to those due to sleep deprivation.

The working hypothesis of the present study was that the SWS impact of sleep deprivation on recovery sleep would be increased by physical exercise.

Two levels of sleep loss were investigated in the present investigation: (Study 1) partial sleep loss resulting from two continuous awake periods of 21 hrs separated by a 3-hr nap and (Study 2) total sleep loss resulting from being continuously awake for 45 hrs. The exercise consisted of walking on a treadmill at a speed which would result in a volunteer expending 30% of maximal aerobic work capacity (30% of VO_2 max).

2. Methods

2.1 Subjects.

Twenty physically fit, well-trained, male U.S. Marine Corps volunteer subjects from a reconnaissance battalion at Camp Pendleton, CA participated in Study 1. The mean age of the volunteers was 20.5$\pm$1.7 years, range 18 to 24 years. An analysis of pre-study questionnaires showed that all had experienced field combat maneuvers, averaging continuous wakefulness of 47 hrs. Subjects in Study 2 consisted of 16 physically fit, well-trained male Marine volunteers from two infantry divisions from Camp Pendleton (mean age of 21.7$\pm$2.4 years).

2.2 Exercise Heart Rate and VO_2 Max Determination.

Assessment of VO_2 max was performed on Monday (Day 1) morning. To determine the heart rate (HR) of each volunteer that was associated with an energy expenditure equal to 30% of their VO_2 max, each subject walked continuously for 1.5 min at combinations of each of the following speeds and grades: 2.1, 2.5, and 3.0 mph at 0% grade; and 2, 4, and 6% grade at 3.0 mph. After these walks, each subject was given a test of VO_2 max. In the VO_2 max test, each subject began running at 5.5 mph, 0% grade. The treadmill speed was increased 0.5 mph each min until the ventilatory equivalent of oxygen (VE/VO_2) began to rise consistently. From this point, the speed was held constant and the grade was increased 2% each min until either the VO_2 did not increase during a period of one minute following an increase in workload, or the subject voluntarily terminated the test. VO_2 max was taken to be the greatest one-minute average of VO_2 ml/kg/min determined during the test. Following the determination of VO_2 max, the initially obtained walking

record was examined to find the workload equating to 30% of VO_2 max. Then, the average heart rate (HR) for that period was calculated and used as the target HR for each subject's subsequent exercise level in the study.

2.3 General Procedures

In Study 1, the subjects were tested for VO_2 max on Monday morning at our Physical Fitness Laboratory and were transported that afternoon to our Ergonomics Laboratory where the main study was conducted. Since the Ergonomics Labaoratory was not equipped with a spirometer when Study 1 was conducted, HR was used to set the physical workload at 30% of the individual VO_2 max. In Study 2, an open-circuit spirometer was used for setting the physical workload at 30% of VO_2 max and also for maintaining physical workload at 30% of VO_2 max during the study.

For sleep EEG, C_3 referred to the linked mastoids (A_1 + A_2) was recorded. C_4 to (A_1 + A_2) was used as a back-up placement. Electrooculograms (EOGs) of the right and left eyes, together with EEG, were recorded on a FM instrumentation tape recorder for later computer analyses .

The ambient temperature was maintained at 21.1 C degrees with a relative humidity of 50%.

Throughout the study, the subjects were allowed to interact freely with the investigators, as well as between themselves, except during periods of performance tests requiring concentration of attention.

Many psychological performance tasks were presented to the subjects, using computer video terminals. The details of results are presented elsewhere (Englund et al., 1983; Naitoh, et al., 1982, 1983; Ryman, Naitoh and Englund, in press).

2.4 Data Collection Schedule (Figure 1)

Monday (Day 1): In the morning, each of the two volunteer subjects were given a graded maximum exercise test to determine maximal oxygen uptake (VO_2 max) with simultaneous measurement of heart rate. In the afternoon, familiarization with the procedures and training on the cognitive and psychological tasks was begun. Each subject was randomly assigned to either the control routine group (C-Gp) or experimental exercise routine group (E-Gp). No sleep polygrams were obtained on Monday night (Sleep 0). However, the subjects slept in the Ergonomics laboratory to adapt to the sleep environment of the lab.

Tuesday (Day 2): For Study 1, volunteer subjects assigned to the exercise routine walked on the treadmill, during the first half-hour of each hour (labelled in Figure 1 as A's), in full combat gear, carrying a 27 kg backpack and rifle,

while performing a visual vigilance task. Walking speed was adjusted to maintain the HR of the exercising individual at a level associated with 30% maximal aerobic work capacity. The control subjects performed the same visual vigilance task, during the session A's, while seated before a computer video terminal.

During the second half of each hour, (i.e., B sessions in Figure 1), oral temperature and blood pressure were measured, and the subjects performed the cognitive and psychological tasks.

Subjects in Study 2 experienced the same procedures, except spirometer readings of expired air of the subjects on the treadmill was used, instead of heart rates, to keep the physical workload during the A sessions at 30% max VO_2.

All subjects had 12 1-hr sessions on this day. The E-group walked on the treadmill for six hours from 0800 to 2045. At the end of the 12th session, the electrodes were applied and polygraphic sleep records (Sleep 1) were obtained from all subjects for 8 hrs (2300-0700)

Wednesday-Thursday (Day 3 - Day 4): The first continuous work period (CW1), consisting of 17 1-hr sessions, began at 0800 Wednesday and ended 0300 Thursday. After CW1, the subjects in Study 1 took a 3-hr nap from 0400 to 0700 (Sleep 2) whereas the subjects in Study 2 remained awake and rested for this period.

Thursday-Friday (Day 4 - Day 5): The subjects in Study 1 were awakened at 0700, given breakfast, and began the second 17-hr continuous work period (CW2), at 0800. The subjects in Study 1 and Study 2 were wired for sleep EEG recordings and went to bed for 8 hrs of "recovery" sleep (Sleep 3) from 0400-1200 Friday.

2.5 Hours of Sleep Loss.

To perform psychological tasks during CW1, all subjects were awakened at 0700 Wednesday and remained awake until 0400 Thursday, a total of 21 continuous waking hours. In the sleep loss literature, the duration of sleep loss is usually equated to that of prior wakefulness (Naitoh, 1975), that is 21-hr sleep loss for the CW1. If, however, their habitual bedtime was assumed to be around 2300, they were awake for five hours beyond their habitual bedtime to perform CW1. Thus, we could argue that they experienced 5 hrs of sleep loss at the end of the CW1, instead of 21 hrs.

All subjects in Study 2 experienced 45-hr sleep deprivation.

2.6 Sleep Measures

2.6.1 Quantitative EEG and ECG Measures. In Study 1, the C_3 EEG was analyzed with a digital computer, using a zero-crossing or period analysis program

on digitized sleep EEG data. The zero-crossing analysis program detected time of occurrence of EEG up-crossings, i.e., time when EEG wave changed from being positive to negative in voltage. The period of one full cycle was the time elapsed between two up-crossings. Slow waves were defined as those which fell within the period from 500 msec (2 Hz) to 2,000 msec (0.5 Hz) with a peak to trough amplitude of at least 10 micro volts. For each slow wave, peak amplitude (the difference between voltage at the peak and voltage at the trough) and time between two up-crossings were measured.

Output of this period analysis was condensed into epochs of one minute length. Two measures of slow wave activity from this analysis were: (1) average peak amplitude in micro volts, and (2) total time, in seconds, occupied by the slow waves per one min epoch (slow wave time). Zero-crossing EEG analysis was not available for sleep data on subjects in Study 2.

Although large body movements during sleep produced extraneously high slow activity in the EEG records, which was indistinguishable from true EEG slow waves, no editing was performed for the zero-cross analysis of sleep EEGs.

2.6.2 Manually Scored Sleep Measures. Sleep records were manually scored, according to criteria of Rechtschaffen and Kales (1968) by the senior author of the present paper. Sleep records were blind-scored. The following sleep variables were extracted from manually staged sleep polygraphic data: (1) sleep latency in min, i.e. the elapsed time from the first stage 1 to the first appearance of stage 2, (2) REM latency, i.e. the elapsed time from the first Stage 2 to the first appearance of stage REM, (3) number of subjects showing Sleep Onset REM Periods, i.e. SOREMPs, (4) total duration of stage 2, (5) total duration of stage 4, (6) total duration of stage REM, (7) total duration of stage SWS, (8) percent SWS or percentage SWS with reference to the sum of time spent in stages 1, 2, 3, 4 and REM and (9) sleep efficiency or a percentage of time spent in stages 1, 2, 3, 4 and REM in reference to the total time spent in bed

The data for one subject in the "No Nap/No Exercise" group were lost due to technical difficulties.

2.7 Statistical Analyses.

Student's independent and correlated sample t-tests (SPSS, 1983) were used to evaluate the differences between the baseline (Sleep 1) and recovery night sleep records (Sleep 3) in four groups of subjects: (1) "Nap and Exercise" (Study 1/E-Gp), (2) "Nap and No Exercise" (Study 1/C-Gp), (3) "No Nap and Exercise" (Study 2/E-Gp), and (4) "No Nap and No Exercise" (Study 2/C-Gp).

The results of sleep staging and computer analysis of Nap (Sleep 2) records,(see Table 1), were not statistically evaluated against Sleep 1 and 3 as we were interested in recovery sleep.

Because multiple comparisons between and within-group differences led to problems of intercorrelations between significance tests and inflated Type I errors, the Dunn-Bonferroni method (Dunn, 1959; Myers, 1972) should be used to control an "experimentwise" error rate (Jacobs, 1976). However, the power of the Dunn-Bonferroni method decreases as the number of significance tests increases (Moses et al., 1977). Thus, we adopted three procedural steps, instead of the Dunn-Bonferroni method, to minimize greater chance of committing a Type II error. With 5 sleep measures, the Dunn-Bonferroni method reduced the accepted level of significance from p=.05 to p=.01.

Step A: We reduced the number of sleep variables from nine to five on the basis of a within-group analysis, a method used by Moses, et al.(1977). A within-group analysis of the subjects in the "Nap/Exercise" routine in Study 1 had significant changes in recovery sleep from baseline sleep in five sleep variables out of the nine examined (to be detailed in the Results section). These changes were caused by the combined effects of partial sleep loss, exercise, and/or circadian variation. Whatever the actual causes of the observed changes in sleep, we believed that these five sleep variables should be examined in detail as they would be sensitive in evaluating the effect of experimental treatments on sleep.

Step B: The exact probability was calculated for each observed t ratio. Given the exact p-value, one could judge the reliability of the observed differences against the Dunn-Bonferroni corrected significance.

Step C: Comparisons were then made between "No-nap Exercise" group and all the other three groups during baseline and also during recovery sleep. The between-group comparisons of baseline sleep data were made to determine that no baseline differences existed for the five sleep variables between the groups. Given no differences in the group's baseline values, recovery sleep comparisons could be assessed.

For the purposes of the present study, the level of significance was determined to be 5% or better, using a two-tail test.

3. Results

3.1 In Study 1, the average HR for the E-group during the treadmill walk was 89.0+$\pm$ 2.9 beats per minute (bpm), very close to the targeted average HR of 90.5+$\pm$

14.3 bpm determined to be necessary to maintain 30% VO_2 max level of exercise. HR data were not analyzed in Study 2 as we obtained spirometric readings for establishing physical workload of the subjects.

3.2 Sleep Stages.

The results and analyses of manually scored sleep stages are shown in Tables 1 and 2.

Table 1 shows that recovery sleep was clearly different from baseline sleep within each group of subjects, with the exception of stage 2 and REM duration. As previously mentioned, five sleep variables of "Nap/Exercise" subjects showed significant changes in recovery sleep. They are starred in Table 1, i.e., shortened sleep and REM latencies (including some SOREMPs), increased stage 4, SWS duration and %SWS. Incidents of SOREMPs during recovery sleep could be attributed more to bedtime shift to early morning hours, than to altered REM need as the results of sleep loss and/or exercise. Table 1 also lists Sleep 2 data to show that subjects did manage to sleep during a period of 3 hrs between baseline and recovery.

Since our subjects were randomly assigned to one of the four groups, and there were no statistically significant pre-existing baseline differences (except sleep latency) between the "No Nap/Exercise" and the others, we compared recovery sleep of subjects in the "No Nap/Exercise" routine with that of all others (see Table 2).

Recovery sleep of subjects in the "No Nap/Exercise" routine (N=8) had significantly longer stage 4 sleep (t=2.56, df=13, p=0.025) and greater SWS% (t=2.42, df=13, p=0.031) than those in the "No Nap/No Exercise" routine (N=7) (Table 2). Table 2 shows also that recovery sleep of subjects in the "No Nap/Exercise" routine had significantly longer stage 4 sleep, but not SWS%, than those in the "Nap/Exercise" (t=2.25, df=13, p=0.033) and those in the "Nap/No Exercise" (t=2.31, df=13, p=0.036) routines.

3.3 EEG Slow Wave Analysis.

The results of EEG slow wave analysis, as shown in Table 3, was limited to Study 1 due to unavailability of the computer to do a zero-crossing analysis in Study 2. For the experimental subjects in the "Nap/Exercise" routine, the average EEG slow wave amplitude during Recovery sleep (Sleep 3) was 6.5 micro volts larger than the amplitude observed during Baseline sleep (Sleep 1). This within- group increase was significant (p=0.015). Comparison of the amplitude increase in the

"Nap/Exercise" routine with that of the "Nap/No Exercise" routine was not significant. Since a similar within-group increase in slow wave amplitude was observed in the "Nap/No Exercise" group, observed changes were most probably due to partial sleep loss, rather than to exercise.

The same statistical evaluation analysis was performed for EEG slow wave time (Table 3). The subjects in the "Nap/Exercise" routine showed a significantly increased EEG slow wave time during Sleep 3 in comparison with Sleep 1 ($p=0.018$). The between-group comparison, "Nap/Exercise" vs. "No-Nap/No Exercise," of the EEG slow wave time was not significant, suggesting again that partial sleep loss, not the exercise, increased SWS time.

4. Discussion

A detailed examination of these between-group differences revealed that subjects in the "Nap/No Exercise" routine had 75.0 minutes of stage 4 during recovery sleep, while subjects from the "Nap/Exercise" routine had 72.7 min long stage 4 in recovery sleep (Table 1). Obviously, whether or not the subjects had exercised on the treadmill did not affect stage 4 duration, as long as the subjects had a 3-hr nap. If, however, subjects did not sleep at all and exercised, i.e., in the "No Nap/Exercise" routine, duration of stage 4 in recovery sleep was 110.1 min, while subjects from the "No Nap/No Exercise" routine had 77.3 min long stage 4 in recovery sleep--a difference of 32.8 min. Thus it appears that the effect of exercise on stage 4 depends on whether subjects experienced total sleep deprivation interacts with exercise.

We suggest that total sleep deprivation interacts with exercise resulting in a greater impact on recovery sleep than total sleep loss alone. This suggestion is not consistent with Webb and Agnew (1973), but partialy supports the findings of Moses et al. (1977). Webb and Agnew (1973) reported that total sleep loss of 48 hours and exercise on a stationary bicycle for 15 min (at 20 km/hr with a 2.5 kg friction load) every other hour for a total of 1 hour per night over two nights did not produce differential recovery effects, i.e. exercise-specific increase in SWS. Moses et al. (1977) deprived subjects of sleep for 40 hrs. Their exercise group bicycled one hour out of every four hours, maintaining a 50% increase in heart rate above the subject's resting heart rate. They found small but nonsignificant increases in %SWS, and reported that moderate exercise slightly and nonsignificantly increased sleep loss effects on recovery sleep.

The conclusion from the present study is based however, on groups of 8 and 7 individuals and a significance level of p=0.03 (two tails), which does not meet the p=0.0017 Dunn-Bonferroni significance criterion required for 30 t-tests (Table 2). Further experimental confirmation is required before we could conclude that a definite increase in stage 4 and %SWS was observed due to exercise only under total sleep deprivation. Quantitative analysis of EEG slow wave activity of totally sleep deprived and exercised subjects would be highly desirable.

Research on the relation between exercise and SWS, without sleep deprivation, during recovery sleep has yielded mixed results. Four papers from two groups of researchers have supported the hypothesis that exercise results in an increased need for SWS (Zolty, Burdick and Adamson, 1973; Shapiro, Griesel, Bartel and Jooste, 1975; Shapiro and Verschoor, 1979; Shapiro, Davis and Daggett, 1981). In these studies, experimental subjects were clearly physically fit and well trained as were the subjects in the present study.

Nine studies have only partially supported the above hypothesis. In one study an increase of SWS was observed only in the early portion of the recovery sleep, while in another study an increase of stage 3 was observed only in the first sleep cycle following afternoon exercise. These nine papers are by Baekeland and Lasky (1966), Horne and Porter (1975), Moses et al. (1977), Griffin and Trinder (1978), Trinder, Paxton, Montgomery, Bowling and Bruck (1979), Bonnet (1980), Buguet, Roussel, Angus, Sabiston and Radomsky (1980), Browman and Cartwright (1980) and Bunnel, Bevier and Horvath (1983). These studies utilized some experimental subjects who were trained and others who were untrained.

Finally, another eight studies have failed to support the hypothesis (Hauri, 1969; Zir, Smith and Parker, 1971; Webb and Agnew, 1973, Adamson, Hunter, Ogunremi, Oswald and Percy-Robb, 1974; Desjardins, Healey and Broughton, 1974; Walker, Floyd, Fein, Cavness, Lualhati, and Feinberg, 1978; Porter and Horne, 1981; Abad and Lucas, 1981).

The absence of effects of exercise on SWS in Study 1 would not necessarily mean that exercise failed to enhance the SWS process. Exercise could increase an intensity dimension of SWS. Torsvall, Akerstedt and Lindbeck (1984) used spectral analysis and observed an increase of total spectral power as well as slow wave activity in sleep EEGs after an extremely strenuous cross-country race. A zero-crossing analysis of sleep EEG in Study 1 did not show the differential increase in slow wave amplitudes and time. Our findings do not agree with Torsvall et al.

(1984), but are consistent with Zepelin et al. (1981) and Walker et al. (1978). Zepelin et al. (1981) observed that bicycle exercise did neither elevate auditory awakening threshold, nor increase EEG amplitude. Walker et al. (1978) examined ten male cross-country/distance runners and another ten matched control male non-runners and found that manually scored sleep stage did not show an increase of absolute amount nor an increased proportion of stages 3 and 4 during sleep after exercise; and that their computer zero-cross analysis of sleep EEGs did not show that a higher level of exercise was accompanied with a higher level of stages 3 and 4.

Our study of those volunteers who napped for 3 hrs (thus partially sleep deprived) and exercised around 30% VO_2 max workload joins a growing number of studies which fail to show exercise-induced SWS increase. However, the present study did find that the impact of total sleep deprivation (45 hrs) in increasing SWS was made greater by physical exercise (30% VO_2 max). This interaction of total sleep loss and exercise has not previously been observed. Thus, exercise seems not to have a simple, direct effect on SWS.

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Table 1 Differences in Sleep Characteristics of Control and Experimental Exercise Groups of Young U. S. Marine Corps Volunteers in a Study of Sustained Operation (Mean ± Standard Deviation)

		Baseline Sleep (Sleep 1)		Nap (Sleep 2)		Recovery Sleep (Sleep 3)	
Sleep Measure	Study	C-Group	E-Group	C-Group	E-Group	C-Group	E-Group
*Latency to Stage 2 (in Min)	#1	14.9± 8.8	16.9±14.1	9.0± 5.9	8.8± 7.1	7.8± 4.2	9.4± 8.5
	#2	9.3± 4.2	27.3±14.2	N/A	N/A	7.2± 5.4	4.6± 3.8
Latency to Stage REM (in Min)	#1	83.7±33.3 (0 SOREMPs)	76.4±26.5 (0 SOREMPs)	36.3±29.2 (4 SOREMPs)	45.7±28.3 (3 SOREMPs)	43.6±29.2 (0 SOREMPs)	63.6±33.7 (0 SOREMPs)
	#2	76.7±17.5 (0 SOREMPs)	88.1±35.4 (0 SOREMPs)	N/A N/A	N/A N/A	54.6±28.8 (1 SOREMPs)	49.5±31.4 (2 SOREMPs)
Duration of Stage 2 (in Min)	#1	176.5±24.5	190.6±14.7	49.1±20.8	59.8±18.5	191.3±25.5	211.6±27.6
	#2	236.0±18.9	201.9±22.7	N/A	N/A	227.9±36.1	206.2±48.0
*Duration of Stage 4 (in Min)	#1	54.9±25.4	53.7±24.7	58.8±21.6	38.7±26.9	75.0±36.3	72.7±39.1
	#2	42.6± 7.9	48.4±13.0	N/A	N/A	77.3±22.8	110.1±26.4
*Duration of REM (in Min)	#1	113.8±22.6	99.9±31.3	36.2±17.8	34.5±11.2	97.3±16.5	77.3±21.0
	#2	111.8±27.0	118.7±21.7	N/A	N/A	105.3±29.5	92.7±55.4
*Duration of SWS (in Min)	#1	88.8±23.9	85.9±26.0	75.3±17.1	66.6±21.4	113.1±33.9	112.2±40.1
	#2	77.6±12.4	77.6±11.2	N/A	N/A	117.6±24.5	140.9±20.7
*% SWS	#1	22.5± 5.9	21.4± 6.4	45.3±10.3	39.9±12.2	27.6± 7.8	27.3± 9.5
	#2	17.5± 2.6	18.0± 2.9	N/A	N/A	25.5± 5.1	31.6± 4.5
Sleep Efficiency (in %)	#1	95.2± 2.4	96.4± 1.8	96.6± 1.8	96.7± 2.1	95.8± 3.4	96.0± 2.4
	#2	97.3± 1.6	94.8± 1.6	N/A	N/A	97.4± 2.3	98.3± 1.0

Table 2 Comparisons of "No Nap/Exercise" Group with All Other Groups during Baseline Sleep and Recovery Sleep.

Comparisons	Baseline Sleep (No Nap/No Ex) vs. (No Nap/Ex)	Recovery Sleep (No Nap/No Ex) vs. (No Nap/Ex)	Baseline Sleep (Nap/Ex) vs. (No Nap/Ex)
Stage 4 (min)	1.Ø2 (Ø.33)	2.56(Ø.Ø25)*	Ø.58(Ø.572)
SWS (min)	-Ø.Ø1 (Ø.99)	1.99(Ø.Ø68)	Ø.9Ø(Ø.384)
SWS (%)	Ø.39 (Ø.7Ø)	2.42(Ø.Ø31)*	1.46(Ø.167)
Stage REM (min)	Ø.55 (Ø.59)	-Ø.54(Ø.6Ø3)	-1.47(Ø.163)
Sleep Latency (min)	3.23 (Ø.Ø1)*	-1.1Ø(Ø.295)	-1.49(Ø.16Ø)

Comparisons	Recovery Sleep (Nap/Ex) vs. (No Nap/Ex)	Baseline Sleep (Nap/No Ex) vs. (No Nap/Ex)	Recovery Sleep (Nap/No Ex) vs. (No Nap/Ex)
Stage 4 (min)	2.35(Ø.Ø33)*	Ø.69(Ø.5Ø1)	2.31(Ø.Ø36)*
SWS (min)	1.93(Ø.Ø75)	1.29(Ø.218)	2.1Ø(Ø.Ø54)
SWS (%)	1.23(Ø.238)	2.11(Ø.Ø53)	1.35(Ø.196)
Stage REM (min)	-Ø.7Ø(Ø.5Ø6)	-Ø.46(Ø.656)	-Ø.21(Ø.838)
Sleep Latency (min)	-1.59(Ø.135)	-2.Ø6(Ø.Ø69)	-1.63(Ø.125)

Student's t (and its 2-tail p-value) for two independent samples was calculated for each of the differences between "No Nap/Ex" group (baseline sleep) and all other groups (baseline sleep), and between "No Nap/Ex" group (recovery sleep) and all other groups (recovery sleep).

Table 3 EEG slow waves during sleep: Slow wave time (Percent EEG "delta" time) and slow wave ampliutde: Mean (Standard Deviation)

	Slow wave amplitude in μV			Slow wave time (Seconds per each minute).		
Subject ID	Sleep 1	Sleep 2	Sleep 3	Sleep 1	Sleep 2	Sleep 3
MR02C	58.5(34.3)	97.1(32.8)	94.5(39.2)	12.0(7.8)	19.6(8.2)	19.7(10.5)
MR03C	70.6(38.7)	88.9(34.6)	78.7(41.2)	24.7(10.2)	27.7(6.5)	26.4(10.4)
MR05C	59.5(25.8)	61.0(6.9)	60.6(29.8)	20.1(10.1)	22.3(3.7)	23.6(10.5)
MR06C	82.5(41.6)	124.9(26.6)	97.7(42.7)	24.5(10.9)	30.4(8.5)	24.9(10.8)
MR07C	57.2(26.0)	69.0(10.7)	60.9(22.8)	17.6(8.0)	18.9(5.6)	17.1(8.3)
MR09C	93.7(40.5)	103.8(25.8)	79.9(28.6)	18.1(12.1)	27.1(6.5)	18.7(9.5)
MR10C	62.5(24.1)	70.1(3.6)	58.8(23.0)	16.6(7.9)	15.1(1.0)	10.9(6.7)
MR11C	60.9(18.2)	----------	79.4(27.2)	14.2(7.3)	----------	20.9(9.8)
Mean	68.2μV	87.8μV	76.3μV	18.5 sec	23.0 sec	20.3 sec
MR02E	62.6(30.7)	84.6(12.9)	70.1(28.8)	19.2(9.5)	26.4(1.9)	23.1(9.0)
MR03E	54.5(20.3)	67.9(4.7)	53.5(17.9)	22.4(9.2)	23.6(3.4)	22.6(10.3)
MR05E	65.6(21.4)	69.7(9.0)	62.7(18.8)	15.7(8.6)	23.0(4.2)	19.8(7.8)
MR06E	81.7(29.9)	81.5(15.3)	82.7(25.0)	22.2(9.6)	22.6(3.8)	26.3(8.2)
MR07E	76.2(40.3)	96.0(9.7)	84.1(33.2)	26.9(12.3)	32.8(3.7)	29.4(13.0)
MR09E	59.3(21.7)	74.6(10.6)	69.7(28.4)	17.1(8.2)	24.1(4.9)	22.9(9.5)
MR10E	44.1(18.2)	61.2(14.4)	58.8(23.0)	16.4(4.6)	13.5(6.1)	16.9(5.1)
MR11E	72.9(32.7)	97.0(18.0)	86.8(39.6)	17.1(10.2)	26.8(3.0)	23.7(11.7)
Mean	64.6μV	79.1μV	71.1μV	19.6%	24.1%	23.1%

C = Control subject: E = Experimental exercise subject.

	MONDAY		TUESDAY		WEDNESDAY		THURSDAY		FRIDAY	
EXPT'L PHASE	Training		Baseline		CW1		CW2		Recovery	
TIME	WA	WB	WA	WB	WA	WB	WA	WB	WA	WB
00 - 01										
01 - 02										
02 - 03										
03 - 04			SLEEP 9		SLEEP 1					
04 - 05										
05 - 06							SLEEP 2		SLEEP 3	
06 - 07										
07 - 08			----- BREAKFAST* -----							
08 - 09			1A	1B	1A	1B	1A	1B		
09 - 10			2A	2B	2A	2B	2A	2B		
10 - 11			3A	3B	3A	3B	3A	3B		
11 - 12			4A	4B	4A	4B	4A	4B		
12 - 13			----- LUNCH -----						LUNCH	
13 - 14			5A	5B	5A	5B	5A	5B		
14 - 15			6A	6B	6A	6B	6A	6B		
15 - 16			7A	7B	7A	7B	7A	7B		
16 - 17			8A	8B	8A	8B	8A	8B		
17 - 18			----- SUPPER -----							
18 - 19			9A	9B	9A	9B	9A	9B		
19 - 20			10A	10B	10A	10B	10A	10B		
20 - 21			11A	11B	11A	11B	11A	11B		
21 - 22			12A	12B	12A	12B	12A	12B		
22 - 23			----- SNACK** -----							
23 - 24	SLEEP 0		SLEEP 1		13A	13B	13A	13B		
					14A	14B	14A	14B		
					15A		15A			

CW = Continuous Work

*Includes time for attachment of ECG electrodes and rifle assembly task.

**Includes time for rifle assembly task.

Figure 1. Data Collection Protocol for Studies 1 and 2.

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Heart Rate	Stage 4 sleep
Nap	VO_2 max
Sleep loss	

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The effect of exercise and sleep loss on sleep was studied in four groups of young, physically fit, well-trained U.S. Marine Corps male volunteer subjects. In the first study, Study 1, ten pairs of Marines were observed. One member of each pair was assigned to an "exercise" routine and walked on a treadmill in full combat gear at a speed that induced an elevated heart rate corresponding to 30% of the individual's VO_2 max. Exercise periods lasted half an hour per hour for two 17-hr long periods of continuous work, designated CW1 and CW2. The two CW episodes were separated by a 3-hr nap. Individuals

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20. Abstract (continued)

assigned the exercise routine spent the second half of each hour in the CW period performing cognitive tasks using a computer terminal. The remaining member of each pair was assigned to a non-exercise, "control" routine. Study 1 consisted of two routines and data on the participants and were grouped as follows: (1) "Nap/Exercise", and (2) "Nap/No Exercise." In a second study, Study 2, eight pairs of Marines participated. In Study 2 the experimental protocol was identical with Study 1 with the exception that neither the exercising nor the control subject were permitted to nap between CW1 and CW2 (7½ Days). The participants in Study 2 were grouped as follows: (1) "No Nap/Exercise" and (2) "No Nap/No Exercise." Our results showed that sleep loss increased manually scored Slow Wave Sleep (SWS) duration, and computer-measured EEG slow wave amplitude, as well as slow wave time during recovery sleep following the CW2. When subjects were totally sleep deprived, exercise increased stage 4 and percent SWS. The observed interaction of sleep loss and exercise on SWS was, however, not strong and would require further experimental confirmation. A working hypothesis of exercise-induced SWS was only partially accepted in this study.